L	Hits	Search Text	DB	Time stamp
Number 1	28	(bachovchin-william\$ or plaut-andrew\$ or drucker-daniel\$).in.	USPAT; US-PGPUB	2003/07/18 10:42
2	42	(demuth-h\$).in.	USPAT; US-PGPUB	10:12 12003/07/18 10:48
3	9901	(514/2,18,19,119,423,626;530/330,331).ccls		; 2003/07/18 10:49
. 4		dp\$liv or dpp\$liv or (dp! or dpp!) adj 'iv! or dipeptidylpeptidase or dipeptidyl	USPAT; US-PGPUB	10.43   2003/07/18   11:21
1 1		adj peptidase or boropro\$ or valboropro\$ or proboropro\$	05 16100	11.21
5		((514/2,18,19,119,423,626;530/330,331).ccl and (dp\$liv or dpp\$liv or (dp! or dpp!)	su\$PAT; US-PGPUB	2003/07/18 10:51
		adj iv! or dipeptidylpeptidase or dipeptidyl adj peptidase or boropro\$ or	†	10.01
6	121859	valboropro\$ or proboropro\$) diabet\$ or glucose or glucagon or glp\$2 or insulin	USPAT; US-PGPUB	12003/07/18 11:21
17	93	'(((514/2,18,19,119,423,626;530/330,331).cc		2003/07/18
:		and (dp\$liv or dpp\$liv or (dp! or dpp!) adj iv! or dipeptidylpeptidase or dipeptidyl adj peptidase or boropro\$ or valboropro\$ or proboropro\$)) and (diabet\$	US-PGPOB	10:52
8	74	or glucose or glucagon or glp\$2 or insufficient (insufficient glp) and (dp\$liv or dpp\$1iv or (dp! or dpp!) adj iv! or dipeptidylpeptidase or	USPAT; US-PGPUB	2003/07/18
		dipeptidyl adj peptidase or boropro\$ or valboropro\$ or proboropro\$)) and (diabet\$ or glucose or glucagon or glp\$2 or insulin)) not (((bachovchin-william\$ or plaut-andrew\$ or drucker-daniel\$).in.) or		
9	237	(dp@mivher\$dppdliv or (dp! or dpp!) adjiv! or dipeptidylpeptidase or dipeptidyladj peptidase or boropro\$ or valboropro\$ or proboropro\$) same (diabet\$ or glucose or glucagon or glp\$2 or insulin)	USPAT; US-PGPUB	12003/07/18 11:09
10	256		EPO; JPO; DERWENT	2003/07/18
11	63463	diabet\$ or glucose or glucagon or glp\$2   or insulin	EPO; JPO; DEPWENT	2003/07/18 11:21
12	108	<pre>(dp\$liv or dpp\$liv or (dp! or dpp!) ad; iv! or dipeptidylpeptidase or dipeptidyl adj peptidase or boropro\$ or valboropro\$ or proboropro\$) and (diabet\$ or glucose</pre>	EPO; JPO; DEPWENT	2003/07/18 11:21
13		or glucagen or glp\$2 or insulin) ((dp\$liv or dpp\$liv or (dp! or dpp!) adj iv! or dipeptidylpeptidase or dipeptidyl adj peptidase or boropro\$ or valboropro\$ or proboropro\$) and (diabet\$ or glucose or glucagen or glp\$2 or insulin)) and @pd<19980203	EPC; JPO; DERWENT	2003/07/18

Chechoel U1 62 65, 68, 69, 613 Je 2 7-18 2003 5/5/10 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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11621283 BIOSIS NO.: 199800403337

Improved glucose tolerance in Zucker fatty rats by oral administration of the dipeptidyl peptidase IV inhibitor isoleucine thiazolidide.

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JOURNAL: Diabetes 47 (8):p1253-1258 Aug., 1998

ISSN: 0012-1797

DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English

ABSTRACT: The hormones glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide (GLP)-1 act on the pancreas to potentiate glucose-induced insulin secretion (enteroinsular axis). These hormones (incretins) are rapidly hydrolyzed by the circulating enzyme dipeptidyl peptidase IV (DP IV) into biologically inactive NH2-terminally truncated fragments. This study describes the effect of inhibiting endogenous DP IV with a specific **DP IV** inhibitor, isoleucine thiazolidide (Ile-thiazolidide), on glucose tolerance and insulin secretion in the obese Zucker rat. In initial studies, the specificity of Ile-thiazolidide as an inhibitor of incretin degradation was determined using matrix-assisted laser desorption/ionization-time of flight mass spectrometry. These results showed that inhibiting DP IV activity with Ile-thiazolidide blocked the formation of NH2-terminally truncated GIP and GLP-1. Oral administration of Ile-thiazolidide resulted in rapid inhibition of circulating DP IV levels by 65% in obese and lean Zucker rats. Suppression of DP IV levels enhanced insulin secretion in both phenotypes with the most dramatic effect occurring in obese animals (150% increase in integrated insulin response vs. 27% increase in lean animals). Ile-thiazolidide treatment improved glucose tolerance in both phenotypes and restored qlucose tolerance to near-normal levels in obese animals. This was attributed to the glucose-lowering actions of increasing the circulating half-lives of the endogenously released incretins GIP and, particularly, GLP-1. This study suggests that drug manipulation of plasma incretin activity by inhibiting the enzyme DP IV is a valid therapeutic approach for lowering glucose

5/5/11 (Item 2 from file: 5)
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11048853 BIOSIS NO.: 199799669998
Improved insulin secretion and oral glucose tolerance after in vivo inhibition of DPP-IV in obese Zucker rats.
AUTHOR: Balkan B; Kwasnik L; Miserendino R; Mone M; Hughes T E; Li L AUTHOR ADDRESS: Sandoz Research Inst., E. Hanover, NJ\*\*USA JOURNAL: Diabetologia 40 (SUPPL. 1):pA131 1997
CONFERENCE/MEETING: 16th International Diabetes Federation Congress Helsinki, Finland July 20-25, 1997